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Guidelines for Surface Water Quality

Vol. 1 Inorganic Chemical Substances

NICKEL

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Abstract

A literature survey was carried out on the toxic effects of nickel and nickel compounds on human health, aquatic life, plants and livestock. The information is summarized in this publication. From it, maximum nickel concentrations in water at which toxic effects will not appear are recommended.

Résumé

On a effectué une étude de la documentation portant sur les effets toxiques du nickel et de composés du nickel sur les humains, les organismes aquatiques, les plantes et le bétail. Ces données sont résumées dans cette publication. En se basant sur ces données, on propose des valeurs des concentrations maximales de nickel dans l'eau auxquelles on n'observe pas d'effets toxiques.

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RECOMMENDED WATER QUALITY OBJECTIVES FOR NICKEL

Uses	Recommended objectives (as total nickel)
Raw public water supply	0.25 mg/L for waters which will be subject to conventional treatment
Aquatic life and wildlife	0.025 mg/L in soft water 0.25 mg/L in water of hardness >150 mg/L as $CaCO_3$
Agricultural water supply	
Livestock watering	5.0 mg/L
Irrigation	0.2 mg/L for continuous use 2.0 mg/L for intermittent use
Recreation and aesthetics	
Recreation	0.2 mg/L
Aesthetics	No recommended limit
Industrial water supplies	
Food processing	0.2 mg/L

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OCCURRENCE, SOURCE, FORMS

Natural Occurrence

Nickel is fairly abundant in the earth's crust, its average concentration being 75 mg/kg. It ranks as the 23rd element in order of abundance and is more abundant than other common metals such as copper, tin or lead (Taylor 1964). Its concentration ranges from 1 mg/kg in sandstone (sedimentary rock) to 160 mg/kg in basalt and gabbro to 2000 mg/kg in peridotite (igneous rocks) (NAS 1975, p. 5ff). In sea water nickel ranks 24th in abundance, with an average concentration of 0.0054 mg/L (Bowen 1966, pp. 19-20).

Nickel concentrations in natural surface waters can go up to approximately 0.100 mg/L, although concentrations of up to 11.0 mg/L have been encountered, especially in mining areas (NAS 1975, p. 8ff). Nickel concentrations (as "extractable") in almost 4800 river water samples collected from various parts of Canada between 1972 and 1977 ranged between less than 0.001 mg/L and approximately 0.3 mg/L. Ninety per cent of these results were less than 0.012 mg/L. The "extractable" nickel concentrations in 360 lake water samples also collected between 1972 and 1977 in various parts of Canada ranged between less than 0.001 mg/L and approximately 0.1 mg/L. Ninety per cent of the results were less than 0.008 mg/L (WQB 1977). Data quoted by Matheson (1975) showed nickel concentrations ranging from 0.002 to 0.004 mg/L in waters from Lakes Erie and Ontario and ranging from non-detectable to 0.028 mg/L in samples of municipal water intakes in the same region.

The concentration of nickel in river sediments is generally much higher than in the overlying waters. Suspended sediments from the Binghamton, New York area contained 150 mg Ni/kg, a concentration 23 000 times higher than the water concentration. In general, the bottom sediments contained 20 mg/kg of nickel, which is approximately the same concentration as that of soil in the region (McDuffie *et al.* 1976). Nickel concentrations in sediment from the Ottawa River were from 1 to 85 mg/kg, and in the Rideau River from non-detectable to 135 mg/kg, with mean concentrations of 26 and 22 mg/kg, respectively (Oliver and Agemian 1974). Much higher values have been found in lake sediments near mining operations (silver, copper) in the Northwest Territories. For example, at some stations in Echo Bay of Great Bear Lake, nickel concentrations of 190 to 1050 mg/kg were found. Similarly, the nickel concentration in the sediment of Ho-Hum Lake was 930 mg/kg (Falk *et al.* 1973, p. 53ff).

Data on drinking water from 233 localities in Canada show that average nickel concentrations are from 0.0029 to 0.0072 mg/L (Neri 1976). Nickel has not been found in most groundwaters (NAS 1975, p. 8ff).

Most nickel is produced from sulphide ores, such as pentlandite $(Fe,Ni)_9S_8$, in which it occurs mainly with iron and copper. This is the ore found in Sudbury (Ontario) at one of the world's largest deposits of nickel. Canada is the world's largest producer of nickel, accounting for 34% of the world total production in 1975. Canada produced 244 782 tonnes in 1975 and 268 526 tonnes in 1974. Consumption of nickel in Canada is small. Of the 268 526 tonnes produced in 1974, 11 567 tonnes was used in Canada and the rest went to exports (Gauvin 1976). Nickel production is concentrated in Ontario (180 929 tonnes in 1975) and Manitoba (63 853 tonnes in 1975). Sulphide ore deposits also occur in South Africa, USSR, and USA (Minnesota). Other types of nickel deposits are the oxide ores, such as garnierite $(Ni,Mg)_6Si_4O_{10}(OH)_8$ or limonite $(Ni,Fe)O(OH).nH_2O$. Although the oxide ores contain the largest proportion of the world's nickel content because they are widely distributed, they account for only one-third of the world's production. The arsenide ores, such as niccolite (NiAs), chloanthite ($NiAs_2$) and nickel glance ($NiAsS$), which occur in Canada, Spain and Germany, are of less importance (Nicholls 1973).

Forms of Nickel

Nickel can form compounds in a series of valence states from -1 to +4. The most common are the Ni(II) compounds. In aqueous solution, nickel commonly shows

only the +2 state. The ion $[Ni(H_2O)_6]^{+2}$ is green and the solutions of Ni(II) have this colour as do most of the hydrated salts.

Nickel forms organometallic compounds. Simple alkyls and aryls of nickel are usually too unstable to exist on their own, whereas the compounds of the form $(R_3P)_2NiX(Ar)$ and $(R_3P)_2Ni(Ar)_2$—where Ar is o-tolyl, o-chlorophenyl, mesityl, etc.; X is a halogen; and R is Et, Ph, etc.—are stable solids with a high melting point (Nicholls 1973).

Nickel (II) halides and other simple salts form a very large number of complexes with ligands which have, in particular, nitrogen or phosphorus donor atoms (Nicholls 1973).

In a study of the Yukon and Amazon Rivers only 2.2 to 2.7% of the total nickel was found in solution as free ions, inorganic and organic complexes. The rest was found adsorbed on suspended materials, in metallic coatings, or incorporated in solid biological materials or in crystalline structures (Gibbs 1973).

In an intensive study of the Susquehanna River (Carpenter *et al.* 1975) it was found that the total nickel concentration (also Mn, Fe, Cd, Cr, Co, Cu and Zn) was correlated with the discharge of the river and with the total suspended solids. This showed that especially in periods of high discharge, most of the metals are found in the suspended materials phase.

Uses of Nickel

The uses of nickel are based on its resistance to corrosion, high strength over a wide temperature range, good alloying properties, and good appearance, i.e. it is silvery white and capable of taking a high polish.

The manufacture of stainless steel, nickel plating, and high-nickel alloys are the main uses of nickel. High-nickel alloys are used in chemical, marine, electronic, nuclear, and aerospace applications. Nickel is also used as a catalyst in industrial processes and in oil refining. Newer applications include nuclear generating plants, gas turbine engines, cryogenic containers, and pollution abatement equipment (Gauvin 1976; Nicholls 1973).

Pathways for Entering the Environment

Nickel enters the environment through two main pathways: (i) weathering of minerals and rocks and (ii) as a result of human activities.

It has been estimated that approximately 160 000 tonnes of nickel enters the environment yearly as a result of the weathering process; around 7% is in the dissolved phase and the rest in sediments (Bertine and Goldberg 1971). Another potential natural source, volcanic activity, has not been studied as a nickel source to the environment, and estimates regarding its contribution to the environment are lacking (NAS 1975, p. 39).

It has been estimated that the human activity that releases the most nickel to the environment is the burning of fossil fuels, i.e. coal and oil. Nickel concentration in crude oil ranges from 0.6 to over 300 mg/kg (Demayo *et al.* 1979). Global oil consumption in 1974 was 2.743 billion tonnes (Skinner 1976). Using an average nickel concentration of 10 mg/kg (Bertine and Goldberg 1971), it can be calculated that the crude oil consumed in 1974 contained approximately 27 000 tonnes of nickel. About 10% of this amount enters the atmosphere (Bertine and Goldberg 1971). Coal contains between 2 and 50 mg Ni/kg. The coal consumption for the generation of energy in 1974 was approximately 1.5 billion tonnes (McMullen 1976). Using an average concentration of 15 mg Ni/kg of coal (Bertine and Goldberg 1971), it can be calculated that the coal burned contained about 22 500 tonnes of nickel. According to Klein *et al.* (1975), in the United States all nickel is retained in the bottom ash and in the fly ash collected in the pollution control equipment. Klein *et al.* (1975) have estimated that globally 10% of the total nickel content of coal is released to the atmosphere. This would amount to about 2250 tonnes per year.

The total world emission of nickel to the atmosphere has been estimated at 5985 tonnes, of which 5151 tonnes was released through the combustion of coal and oil (Sittig 1975, p. 100). This last value is very close to the value estimated here of 4950 tonnes.

Various other human activities release nickel to the environment. In New York City, 62% of the nickel reaching the city's 12 major wastewater treatment plants originates in the electroplating industry. The rest has its origin in a variety of other industrial and non-industrial activities. Nickel has been found, for example, in the wastewaters from industrial activities such as fur dressing and dyeing, bakery, soft drinks and flavouring syrup, and ice cream. Urban runoff and residential wastewaters also contained nickel (Klein *et al.* 1974). The human activities which have been identified as the major contributors to the release of nickel in the environment are mining and concentration of nickel ores, smelting and refining of concentrates, melting and casting of alloys, and plating industries. Typical nickel concentration ranges in wastewaters are 0.46 to 3.4 mg/L in acid mine drainage and 2 to 900 mg/L in the rinse waters

of plating plants (Sittig 1975, p. 104). No estimates of the global amounts of nickel released to the environment by such industries could be found in the literature.

Recent joint Canadian and United States studies to determine pollutant inputs to Lake Superior and Lake Huron via atmospheric fallout have estimated yearly nickel loading to Lake Superior at 120 tonnes and to Lake Huron at 210 tonnes (Swanson 1976).

Thus, on a global scale it would appear that the amount of nickel entering the environment during the natural cycle is much larger than the amount resulting from human activities.

RATIONALE

Raw Public Water Supplies

Nickel in the Human Body

The main route by which nickel enters the human body is oral ingestion via food and drinking water.

Schroeder *et al.* (1962) have estimated that depending on the composition of the diet, the oral nickel intake can vary between 0.003 and 0.9 mg per day and that the usual intake is between 0.3 and 0.6 mg per day. Stainless steel kitchen and flatware is another source of nickel intake. It has been shown that nickel solubilization by detergents and sweat occurs (Katz and Samitz 1975).

Breathing also contributes to the intake of nickel into the body. The amount of nickel inhaled depends on the nickel concentration in the air and this can vary from nil to values above 1 $\mu g/m^3$ in locations near nickel mining and processing, such as Sudbury, Ontario (NAS 1975, p. 26). Considering that a person inhales around 20 m^3 of air in 24 hours, the daily intake of nickel via the respiratory tract could reach a high of 0.02 to 0.03 mg. The amount of nickel in cigarettes has been estimated at 0.002 to 0.006 mg per cigarette, of which 10 to 20% is released in smoke (NAS 1975, p. 179). Thus, a one pack per day smoker can inhale, approximately, an additional 0.004 to 0.025 mg Ni/day.

Most of the ingested nickel is eliminated very rapidly via feces and urine. It has been estimated that only 3 to 6% of the nickel content of an ordinary diet is absorbed. Several studies on the nickel level in human urine found different ranges. The overall range was approximately 0.001 to 0.081 mg/L and the averages were between 0.01 and 0.02 mg/L (Underwood 1971, p. 171). Another study (McNeely *et al.* 1972) gave an average of 0.0072 ± 0.0039 mg/L (or 0.0079 ± 0.0037 mg/day) for nickel concentration in urine in a series of samples from the population of Sudbury, Ontario, a nickel mining and refining region. The urine excretion of nickel from a comparable group from a non-nickel producing region (Hartford, Conn.) averaged 0.0025 ± 0.0014 mg/day. Levels of nickel in blood serum were also different between the two population groups: 0.0046 ± 0.0014 mg/L for the group from Sudbury and 0.0026 ± 0.0010 mg/L for the group from Hartford, Conn.

The fecal excretion of nickel, 0.08 to 0.54 mg/day (average 0.25 ± 0.126 mg/day), for a group from Hartford, Conn., was approximately 100 times greater than the urinary excretion (Horak and Sunderman 1973). The normal body content of nickel in humans is approximately 10 mg with a wide range of values (International Commission on Radiological Protection 1975, p. 381). Nickel is present in relatively higher concentrations in the lungs, pancreas, adrenal glands, brain (0.43 mg/kg), teeth, bone (108-111 mg/kg in bone ash), kidney, heart, aorta (6 mg/kg), and skin (0.38 mg/kg) than in the testes, ovaries, intestine, blood (0.025-0.35 mg/L), serum (0.0022-0.0553 mg/L), plasma (0.002-0.023 mg/L), and muscle (Nielsen 1971). It has also been shown that nickel is present in human fetal tissues and therefore been concluded that it can pass the placental barrier (NAS 1975, p. 89; Creason *et al.* 1976). In fact, nickel in the blood of embryos reaches its highest level at 12 to 25 weeks, with the newborn still having higher levels than infants (Nielsen 1971).

Nickel is considered one of the newer essential trace elements (Mertz 1974). A good review on the essentiality of nickel was written by Nielsen (1971).

Data on nickel deficiencies are very sparse. From studies with ducks, rats and swine, nickel appears to be involved in lipid metabolism and its deficiency produces changes in the morphology and function of the liver, discolouration and dermatitis of the skin, and some impairment of reproductive performance (Mertz 1974). Patients with liver cirrhosis have shown decreased nickel concentrations in serum. On the other hand, patients with acute myocardial infarction have shown increased concentrations of nickel in the serum (Blomberg *et al.* 1977). If animal results can be extrapolated to man, then the dietary requirement is probably 0.050 to 0.080 mg/kg of diet. Diets high in food of animal origin and fats can be nickel deficient (Nielsen and Sandstead 1974).

Although its physiological role is starting to unravel, the exact mechanism of nickel action is still unclear. Nickel is consistently present in RNA; it is bound to several biological substances such as amino acids, proteins (keratin,

insulin), serum albumins; activates several enzyme systems (arginase, carboxylase, trypsin, acetyl coenzyme A synthetase); and in powdered metallic form, dissolves in horse serum or homogenates of rat tissues and is bound to small diffusible molecules (Blomberg *et al.* 1977; Underwood 1971, p. 172ff; Callan and Sunderman 1973).

Toxicity of Nickel

The literature on nickel toxicity consists mainly of reports of industrial exposure to dust and electroplating materials. Dermatitis known as "nickel itch" and nasal-sinus and lung cancer are among the diseases connected with this type of exposure (Horvath 1976; Falk 1974).

The nickel ingested orally has apparently very low toxicity. Data from animal studies (rats, mice, chicks, etc.) quoted by Underwood (1971, p. 174) and by the National Academy of Sciences (NAS 1975, p. 98) showed that rat and monkey diets containing up to 1000 mg Ni/kg did not adversely affect the respective animals. Data quoted by NAS (1975, p. 100ff) showed that the lethal and toxic doses for nickel and nickel compounds depended on the nature of the nickel compound, route of administration and animal species.

For oral ingestion, the LD_{50} varied from 5 mg/kg (nickel metal in guinea pig) to approximately 520 mg/kg (administered as $Ni(NO_3)_2$ to rats) (NAS 1975, pp. 100-101). Mice tolerated nickel acetate in their drinking water at concentrations of 5 mg/L over their lifetime (NAS 1975, p. 98).

Data from Sudbury, Ontario, a nickel-producing region, showed that the nickel concentration in the municipal tap water ranged between 0.141 and 0.246 mg/L (average 0.200 mg/L). No adverse effects have been observed in the population (McNeely *et al.* 1972).

Injection of Ni(II) in rodents showed that it accumulated in the kidney and pituitary gland. The secretion of prolactin by the anterior pituitary gland was inhibited (Blomberg *et al.* 1977, p. 16). Administration of Ni(II) to animals also interfered with the glucose metabolism in experimental animals. The resulting hyperglycemia could have been produced either by an increase in the secretion of glucagon and damaged α-cells in the pancreas or by a decrease in insulin concentration produced by the β-cells of the pancreas. It is also possible that nickel interferes with the immunological system and neuromuscular transmission (Blomberg *et al.* 1977, pp. 17-18).

Of the nickel compounds, nickel carbonyl, a volatile liquid that is an intermediate product in some nickel refining processes and a compound being used in several other industries, is particularly toxic to humans. Accidental inhalation is the usual cause of nickel carbonyl poisoning. The literature is well documented on this aspect of nickel toxicity and a good review has been published (NAS 1975, p. 113ff).

Recommended Objective for Raw Public Water Supplies

Data obtained from animals show that nickel is one of the essential elements of life. The animal data also show that its oral toxicity is very low, that it does not accumulate in tissues, and that it is eliminated via the feces and urine. There is also evidence for homeostatic regulation of nickel metabolism (NAS 1975, p. 90).

Evidence showed that nickel salts ingested orally affected the reproductive system of rats. For example, the average litter size declined, the offspring mortality increased, and fewer males than normal were born to rats given drinking water containing 5 mg Ni/L continuously over three generations (Schroeder and Mitchener 1971).

Raw water supplies in Canada generally contain low concentrations of nickel. In regions where nickel concentrations are relatively high, e.g. Sudbury, Ontario, no adverse effects have been observed so far in the general population. Nickel-containing vessels, recipients, etc. are used in the food industry or at home for food preparation. Results from animal studies show that nickel is both an essential element and a potentially toxic one when ingested in large amounts. Canadian drinking water standards do not give a limit for nickel.

Biological treatment of wastewaters removes 15 to 40% of the nickel in the influent, the percentage removal becoming smaller as the concentration in the influent decreases. Data quoted by Page (1974, p. 21) showed that with a feed concentration of 0.15 mg/L, the effluent from biological filters had a concentration of 0.08 mg/L. Improved removal can be obtained by physical-chemical systems. Thus, a lime precipitation-activated carbon system can remove 99.5% at an influent concentration of 5 mg Ni/kg (Cohen 1977).

Accepting 5 mg/L as the minimum toxic level, using a safety factor of 25, and assuming at least 20% removal of nickel through conventional treatment, the nickel concentration in raw public water supplies should not be greater than 0.25 mg/L.

Aquatic Life and Wildlife

Aquatic Life–Levels in Water and Organisms

There is recent evidence that nickel is essential to some physiological processes, but its role is not yet clearly understood. Previously, nickel was not regarded as an essential element (see section "Raw Public Water Supplies"). It has been shown to be highly toxic to some aquatic organisms. Its synergistic reactions with copper must be taken into account when considering levels in natural waters.

Accumulation of Nickel by Aquatic Organisms

A number of authors have studied the uptake of nickel by freshwater algae and macrophytes. The concentration factor varied quite widely from 120 in *Sagittaria sagittifolia* and 550 in *Nuphar luteum* (Dietz 1973) to 11 429 in *Potamogeton* sp. and 19 667 in algal periphyton. Studies with freshwater fauna indicated the absence of biomagnification through the food web, as concentration factors for zooplankton, clams and fish were 643, 262 and 329 respectively (Hutchinson *et al.* 1975). Annelida (11 mg/kg) contained more nickel than other members of the food web, and carnivorous fish (0.12 mg/kg) contained less than omnivorous fish (0.17 mg/kg) (Mathis and Cummings 1973). Nickel content of fish in the Northwest Territories was close to the detection limit of 0.1 mg/kg dry weight. The range of nickel levels was quite wide in invertebrates (4.0 to 30.8 mg/kg dry weight) and in plankton (51 to 110 mg/kg) (AOSERP 1977).

Toxicity to Aquatic Life

Algae–Studies using algae from heavily contaminated waters have indicated that algae become tolerant to nickel. Growth continued in water containing levels of nickel that were high enough to be lethal to algae previously unexposed to the metal.

The growth of *Chlorella* sp. and *Scenedesmus* sp. that had been cultured from laboratory strains was inhibited by 0.5 mg/L. Members of the same species collected from nickel-contaminated lakes in the Sudbury region (up to 4.5 mg Ni/L) were more tolerant to the metal and were still growing when 3.0 mg/L was present in the test water, though an effect was evidenced at 1.5 mg/L as a decrease in growth occurred. The level of no effect on the laboratory-cultured strains was 0.250 mg/L (Stokes *et al.* 1973).

Copper and nickel in combination were synergistic in their toxic effects on algae. *Haematococcus* sp. growth was reduced by 17% of the control growth by 0.05 mg Ni/L alone. The same concentration of copper alone reduced the growth by 22%. In combination, the growth was reduced by 82% of the control growth. *Chlorella* sp. reacted similarly to *Haematococcus* sp. (Hutchinson 1973). Hutchinson and Czyrska (1975) showed that part of the synergistic effect seemed to relate directly to metal uptake, as more nickel was taken up by *Lemna valdiviana* when copper was present than when nickel was alone. In the presence of nickel alone, there was an initial rapid uptake with a levelling off and most of the nickel was adsorbed onto the cell. Copper on the other hand, entered the cell at a steady rate; the pattern of nickel uptake changed when copper was present and it entered the cell at a steady rate as did the copper.

Microinvertebrates–Andrivon (1974) studied the effects of nickel on *Paramecium caudatum* (Protozoa). The movement of cilia was prevented by 0.08 mg/L. This effect was decreased by the presence of K^+, Na^+ and Mg^{++} ions. Although it was thought that antagonism of these ions to nickel occurred at the cell membrane and not at the ciliary mechanism, the mechanism of nickel toxicity inhibition was not understood.

The acutely toxic levels (48-h LC_{50}) of nickel to three species of zooplankton–*Cyclops abyssorum prealpinus* (Einsle), *Eudiaptomus padanus padanus* (Burkhard) and *Daphnia hyalina*–was given as 150, 3.6 and 1.9 mg/L respectively (Baudouin and Scoppa 1974).

There was a 16% impairment of reproduction in *Daphnia magna* when it was exposed to 0.03 mg/L for 3 weeks. The LC_{50} for 3 weeks was 0.130 mg/L in soft water (44 mg/L as $CaCO_3$). There was no significant difference between the levels of nickel affecting survival (LC_{50}) and causing 50% reproductive impairment over a period of 3 weeks at 0.130 and 0.095 mg/L, respectively (Biesinger and Christensen 1972).

Macroinvertebrates–The toxicity of nickel to macroinvertebrates varied with the species. The 96-h LC_{50} for a stonefly (*Acroneuria* sp.) was 33.5 mg/L and for a mayfly (*Ephemerella* sp.) was 4.0 mg/L in soft water (44 mg/L as $CaCO_3$) (Warnick and Bell 1969).

Fish–The toxicity to rainbow trout of nickel in combination with zinc, copper and phenol indicated that the combined toxicity could be predicted by the summation of the individual toxicities. The 48-h LC_{50} for nickel was 32.0 mg/L at a water hardness of 240 mg/L as $CaCO_3$ (Brown and Dalton 1970). Synergism was not indicated, which is in contrast with the results when two of the metals, copper and nickel, were present in algal culture media (Hutchinson 1973; Stokes 1975).

There was no significant effect on the reproduction of fathead minnows at 0.38 mg/L (hardness of 210 mg/L as $CaCO_3$), but there was a significant reduction in the number of eggs and in hatching success at 0.73 mg Ni/L (Pickering 1974).

Factors Affecting Nickel Toxicity

An increase in pH decreased the toxicity of nickel to *Chlorella sorokiniana* (phytoplankton) (Moshe *et al.* 1972). Hutchinson and Stokes (1975) showed that nickel was less soluble and hence less toxic at higher pH.

A large percentage of nickel is removed from solution by the presence of suspended solids (see p. 2). In areas of Canada where there are large amounts of suspended solids and other material which would adsorb nickel, the toxicity of the metal would be greatly reduced.

The hardness of water also affected the toxicity of nickel. The 96-H LC_{50} values for fathead minnow in soft water (20 mg/L as $CaCO_3$) were 4.58 and 5.18 mg/L, but in hard water (210 mg/L as $CaCO_3$) the values were 25.0 and 28.0 mg/L. In very hard water (360 mg/L as $CaCO_3$) the 96-h LC_{50} was 42.4 mg/L (Pickering and Henderson 1966; Pickering 1974).

Recommended Objective for Aquatic Life

The U.S. Environmental Protection Agency *Quality Criteria for Water* (EPA 1976) recommended that 0.01 of the 96-h LC_{50} for the species of concern should be used as the maximum level of nickel allowed in water. The International Joint Commission (IJC 1976) recommended a level of 0.025 mg Ni/L.

Little information exists on the sublethal toxicity to aquatic fauna of nickel, which is not as toxic as some other metals, for example, copper and zinc.

Algae, the basis of the food web, were susceptible to concentrations of nickel above 0.25 mg/L. Macroinvertebrates varied in their susceptibility, but *Daphnia magna*, a representative of the zooplankton component of the food web, was affected in its reproductive cycle by 0.030 mg/L.

Fathead minnow reproduction did not appear to be significantly affected by 0.38 mg/L. The acute toxicities for minnow and rainbow trout were similar in hard water; toxicity was greater in soft water.

There is inadequate information on sublethal effects for fish. An application factor of 0.01 (*Guidelines for Surface Water Quality*, Vol. 1, Preamble, p. 5) applied to the 96-h LC_{50} for fathead minnow in soft water of 4.58 mg/L indicated a maximum level to protect fish of 0.045 mg Ni/L. This is higher than levels which have been shown to effect some zooplankters in similar water hardness (*D. magna* 0.03 mg/L). The level recommended in this document for soft water is the same as that recommended by the International Joint Commission, i.e. 0.025 mg Ni/L.

The recommended level of nickel for aquatic life in hard water (>150 mg/L as $CaCO_3$), and in water with high levels of suspended solids and other substances which would remove nickel for solution, is 0.25 mg/L (0.01 x 96-h LC_{50} of 25 mg/L).

Where copper is known to be present, the possibility of synergism must be taken into account. Insufficient experimental data was found in the literature to enable a maximum concentration for nickel, in the presence of copper, to be recommended. Site-specific studies may have to be done to determine the safe level of nickel in water which contains copper.

Wildlife

Very little information was found in the literature on the effects of nickel on wildlife, and the level suggested for aquatic life should safeguard birds and mammals.

Water Supply for Livestock

Essentiality of Nickel in Animal Nutrition

As mentioned in the "Nickel in the Human Body" section, relatively recent experimental data showed that nickel might be one of the essential elements in human, animal and plant nutrition. In fact, among the elements of the 4th row of the periodic table from vanadium (atomic number 23) to zinc (atomic number 30), nickel (atomic number 28) was the only one not considered essential (Nielsen and Ollerich 1974).

Chicks maintained on a nickel-deficient diet showed pigmentation changes and dermatitis of the shank skin, thicker legs with slightly swollen hocks, less friable liver and an enhanced accumulation of a trace dose of ^{63}Ni in the liver, bone and aorta. It has also been shown that nickel deficiency in chicks resulted in suboptimal liver function, consisting of ultrastructural degeneration in the hepatocytes, decreased oxygen uptake of liver homogenates, decreased phospholipid levels and increased total lipid content of the liver (Nielsen and Ollerich 1974). The

nickel-deficient diet contained 0.003 to 0.004 mg Ni/kg. It was concluded that a diet which provides the required amount of nickel should contain 0.050 to 0.080 mg/kg.

In rats nickel apparently affected reproduction, as a significant number of dead pups occurred with first- and second- generation nickel-deficient females (15 and 19% vs. 0 and 10% in the control group, respectively). Also, the third- generation nickel-deficient litters were less active than the control group. Changes in the liver structure and its physiological activity were also observed. It has been postulated that nickel has a role in RNA metabolism (Nielsen *et al.* 1974). Effects on reproduction were observed in pigs fed on a nickel-deficient diet, as a higher percentage (65%) of baby pigs from sows on a nickel-supplemented diet (10 mg/kg) were still alive on their 28th day compared with only 37% of the baby pigs from sows on a low nickel diet (0.1 mg/kg). Also, male and female pigs and goats fed on the low nickel diet had a significant decrease in their rate of weight gain and developed more slowly than the group fed on a diet containing 10 mg/kg of nickel. A decrease in the calcium content of the bones was apparent in the low nickel diet group. Epithelial changes, such as scaly and crusty skin, were observed in adults as well as baby pigs and young goats (Anke *et al.* 1974).

Nickel Toxicity to Animals

Nickel ingested orally has low toxicity, and nickel metal is relatively non-toxic. Dogs tolerated 1000 to 3000 mg/kg without any obvious effect. Dogs and cats tolerated daily doses of 4 to 12 mg/kg for 200 days without ill effects (Stokinger 1963). Nickel salts were also tolerated well by rodents when administered orally. Some of these data were quoted in the section on raw public water supplies.

Toxic effects, such as reduced food intake and growth suppression, were observed in male Holstein calves fed on a diet supplemented with 1000 mg Ni/kg (as $NiCO_3$) over an 8-week period. The average daily intake of nickel was 1557 mg. No such effects were observed when the diet was supplemented with 250 mg/kg or less. The average daily intake of nickel for the diet supplemented with 250 mg/kg was 1281 mg, almost equal to the daily intake for the diet containing 1000 mg/kg. The tissue distribution of nickel changed drastically in the calves receiving a diet supplemented with 1000 mg/kg, as if this diet overcame any existing homeostatic control mechanism even though the total daily nickel intake at this level was not greatly different from that on the 250 mg/kg level (O'Dell *et al.* 1971). In another experiment with male dairy calves the feed intake and growth rate were slightly retarded by the diet containing 250 mg Ni/kg. Animals receiving 1000 mg/kg exhibited an immediate aversion for it and did not adapt with time. The growth suppression effect of the 1000 mg Ni/kg diet disappeared when the animals were fed the basal diet containing 0.9 mg/kg during a recovery period (O'Dell *et al.* 1970a).

No significant effect on milk production, milk composition, animal health or feed consumption was observed when lactating cows were fed on a diet supplemented with 50 and 250 mg Ni/kg, as $NiCO_3$. All of the milk samples from the cows receiving 250 mg/kg contained less than 0.1 mg/L, the detection limit of the analytical method used (O'Dell *et al.* 1970b).

Nickel, as acetate or sulphate, at the 300 mg/kg or above level significantly depressed chick growth (Weber and Reid 1968). Nickel chloride administered orally to young male rabbits at 0.5 mg/day over 5 months decreased liver glycogen, increased muscle glycogen, and produced prolonged hyperglycemia after a galactose load (Gordynya 1969).

Recommended Objective for Water Supply for Livestock

The available data suggest that in low quantities, nickel is an essential element in animal nutrition. Ingested orally, nickel is non-toxic in moderate amounts, but nickel salts in concentrations above 300 mg/kg of diet lead to some toxic effects such as growth reduction.

Presumably because nickel does not present a problem, the Australian water quality criteria (Hart 1974) do not recommend a limit for it in the section on "Livestock Water Supplies". Similarly, the National Academy of Sciences in the United States does not deal with nickel in the section on "Water for livestock enterprises" in their book on water quality criteria (NAS/NAE 1973).

Based on the published results, concentrations of 5 mg/L or lower should be safe for the water supply for livestock.

Water for Irrigation

Nickel in Soils and Plants

Nickel is widespread in the earth's crust and can be found in all soils. The concentration depends on the type of soil and its location. The "normal" concentrations range between 5 and 500 mg/kg. Soils derived from sandstones, limestones or acid igneous rocks contain less than 50 mg/kg, whereas those derived from argillaceous sediments or basic igneous rocks contain up to 500 mg/kg or more. Although not widespread, soils derived from serpentine rocks can

contain up to 5000 mg Ni/kg (Vanselow 1966). The soils in Ontario had an average nickel concentration of 15.9 mg/kg and a range of 1.3 to 119 mg/kg. Soils close to smelter operations had much higher values (up to 6560 mg/kg). Sandy soils had the lowest average, 7.6 mg/kg, and clays and organic soils, the highest averages with values of 27.8 and 28.6 mg/kg, respectively (Frank *et al.* 1976; Hutchinson and Whitby 1973).

Nickel concentrations in plant tissues are proportional to the nickel concentrations in soils and they appear to be closely correlated to the "available" nickel in soil. The "available" (also called extractable or exchangeable) nickel is measured by extracting the soil sample with acetic acid, water, acetic acid and ammonium acetate, HCl, EDTA, etc. (Welch and Cary 1975; Vanselow 1966; Page 1974, p. 27; Mitchell *et al.* 1978). An experiment carried out in Sweden (Andersson and Nilsson 1972) showed that the nickel added to the background level of the soil, in this case by sewage sludge fertilizer, was more readily available to the vegetation (crop not specified) than the background nickel. The soil and plant background nickel concentrations were 28.2 and 4.9 mg/kg (dry basis), respectively. After addition of the sludge the nickel concentrations became 43.3 mg/kg and 9.2 mg/kg, representing increases of 53.5 and 87.8% respectively. It seems that in sewage sludge a higher percentage of the element is in an extractable form than in the soil itself. In some sludges from Edmonton and Lethbridge, Alberta, the "extractable" nickel varied between 14 and 30% depending on the sludge and, mainly, on the acid used to measure the extractable fraction. The percentages were higher when 1 *N* HCl was used for extraction than when 0.5 *N* acetic acid was used (Dudas and Pawluk 1975). The "extractable" fraction in soil is commonly much less than 10% depending on the extractant and the type of soil, and very seldom does it exceed 1 mg/kg (Vanselow 1966; Hutchinson and Whitby 1973). Trace elements such as nickel exhibit high affinity for soil organic matter, forming insoluble and soluble complexes with it. A trace element brought into contact with soil by irrigation water, land disposal of sewage sludge, etc., undergoes chemical and biological transformations. A sizeable portion, however, might remain essentially unchanged. Most probably that part which is not complexed with organic matter exists in soil solutions predominantly as divalent cations (Ni, Ba, Cu, Zn, etc.). In anaerobic soils, solubilities are limited by sulphide precipitation. In the pH range common to soils, 5 to 8.5, concentrations of the cationic trace element do not exceed 0.25 mg/L (Page 1974, p. 63ff).

It was also observed that the nickel (also, Cu, Zn and Cd) availability to plants was greater in acid than in calcareous soils and that the difference between the two types of soil increased with the amount of nickel added to the soil (as sewage sludge containing 34 mg Ni/kg enriched as needed with $NiSO_4$) (Mitchell *et al.* 1978). When no nickel was added to the soil, the water extracts of an acid and of a calcareous soil had equal concentrations of 0.3 mg Ni/L. When the two soils were treated with 80 mg Ni/kg of soil (as sewage sludge containing 34 mg Ni/kg enriched as needed with $NiSO_4$), the water extract of the acid soil had a concentration of 22 mg Ni/L, while the nickel concentration in the water extract of the calcareous soil was only 2.2 mg/L. At a treatment of 640 mg Ni/kg of soil the water extract of the acid soil had a concentration of 336 mg Ni/L, whereas that of the calcareous soil had a nickel concentration of 9.3 mg/L (Mitchell *et al.* 1978).

Several vegetables grown on soil treated with sludge over a 19-year period showed increased levels of nickel (and other metals) over control crops but no toxic effects. The rate of sludge application was 66 tonnes per hectare per year. Assuming that the sludge is mixed in the top 15 cm of soil, that soil density is 1330 kg/m^3 (Page 1974, p. 72) and that nickel concentration (extracted by 0.5 *N* acetic acid) in the sludge is 50 mg/kg (Page 1974, pp. 13, 34, 57), the nickel added to the soil over the 19-year period would have amounted to approximately 30 mg/kg. The soil concentration at the 17-year mark was 18 mg/kg (nickel extracted by 0.5 *N* acetic acid), whereas the untreated lot had a concentration of 4.2 mg/kg. Six years after the treatment had been stopped, the concentration in the treated soil dropped to 8.1 mg/kg and the concentration in the untreated soil was unchanged (LeRiche 1968). Although not directly comparable because of different experimental methods, LeRiche's results agree with those of Andersson and Nilsson (1972) quoted above. In their work, the soil nickel concentration (extractable by 2 *N* HCl) increased from 28.2 to 43.3 mg/kg (dry basis) after 12 years of treatment. During this period a total of approximately 2.8 mg of nickel per kilogram of soil (wet basis) had been applied. Unfortunately it was not possible, from the available data, to convert to a dry basis so that a comparison could be made of the amounts applied to the soil with those retained in the soil. It has been estimated that 0.1 to 10% of the amount applied is removed with the crop products, and additional losses occur by leaching and possibly by erosion.

The Effect of Nickel on Plants

In the last several years evidence that nickel is essential for animal life has begun to accumulate (see sections on "Nickel in the Human Body" and "Essentiality of Nickel in Animal Nutrition"). Evidence on nickel essentiality to plants is still sparse.

An indication that small amounts of nickel might be beneficial to plants can be found, for example, in a

paper by Webber (1972). Oat plants were grown on soil mixed with sludge that had very low levels of nickel and to which nickel sulphate was added to give a concentration of 12.5 mg Ni/kg. These plants had a greater mean height than the control plants. The controls were grown on a soil mixed with the same sludge but to which no nickel was added. At nickel concentrations of 25 mg/kg, toxic effects appeared. Russian data, mainly quoted by Nielsen (1971), showed that small amounts of nickel resulted in improved growth and yields of asparagus, lettuce, peas, oats, grapes, rice, cotton and potatoes. It also increased vitamin P and C production in several vegetables and the production of carotene and chlorophyll in corn and sugar beets.

Recently Dixon *et al.* (1975) have shown that jack bean urease is a nickel metalloenzyme (2.0 ± 0.3 g-atoms of nickel per 105 000 g of enzyme) and that other ureases may also be nickel metalloenzymes.

Nickel concentrations that are toxic to plants vary in magnitude according to plant species. In the early stages of nickel poisoning there are no definite symptoms, only a dwarfing or repression of growth. More advanced stages produce chlorosis, followed by necrosis and death of the plant (Vanselow 1966). The "normal" range for nickel concentration in the edible parts of plants varies between 0.01 mg/kg (tomato fruit) and approximately 35 mg/kg (wheat grain). If the non-edible parts of the plant are considered, the concentration can be higher: up to 56 mg/kg in various grass species and 340 mg/kg in oat tops (Vanselow 1966).

Oat plants grown in nutrient solution showed slight symptoms of nickel toxicity at a concentration of 1 mg/L. At a nickel concentration of 2.5 mg/L, the symptoms became moderate and at 5 mg/L, heavy. The concentration in the leaf tissue (dry basis) increased from 3.8 mg/kg (control) to 50 mg/kg at 1 mg Ni/L in nutrient solution, 153 mg/kg at 2.5 mg Ni/L, and 196 mg/kg at 5 mg Ni/L (Hunter and Vergnano 1953). The yield of oat and mustard grown in pots to which nickel had been added (as nickel sulphate) decreased at added concentrations of 50 mg Ni/kg of soil and up. The mean height of the oat plants decreased when 25 mg/kg was added in the form of sludge compared with plants grown in soil mixed with a sludge with very low levels of nickel (Webber 1972). In the same study Webber (1972) also showed that the toxic effects of nickel were greater at pH 5.7 than at pH 6.7. This agrees with the explanation that the "extractable" nickel is the fraction available to the plant. A yield reduction of 25% was reported in lettuce when approximately 75 mg Ni/kg was added to acid soil. The same yield reduction in a calcareous soil required an addition of approximately 270 mg Ni/kg of soil. As the amounts of nickel added to the soils (as sewage sludge containing 34 mg Ni/kg and then enriched with $NiSO_4$) increased, the yields decreased, reaching for example only 25% of the control yield at added nickel concentrations of approximately 170 mg/kg of acid soil and 570 mg/kg of calcareous soil (Mitchell *et al.* 1978). The yield reductions were also dependent on the plant species. In the same experiments a 25% yield reduction in the wheat grain was obtained at an added nickel concentration of approximately 110 mg/kg of acid soil and 230 mg/kg of calcareous soil. Other similar results were quoted by Page (1974, p. 39ff).

Corn and sunflower plants grown in hydroponic solutions showed decreased net photosynthesis and rates of transpiration, although the total biomass of the roots and plant tops was not significantly different from those of the controls. The nickel concentrations ranged from 1 to 10 mg/L. Net photosynthesis was reduced by 5% to 25% compared with that of the controls, and the reduction was proportional to the accumulation of the metal in the leaves. The amount of nickel accumulated in the leaves ranged between 1% for corn and 5% for sunflower, approximately, of the solution concentration. The rate of transpiration was affected to approximately the same extent as the net photosynthesis (Carlson *et al.* 1975). Corn grown on soil treated with $NiCl_2$ at concentrations of 220 mg Ni/kg was not significantly affected. Apparently nickel in solution is more readily available for plant uptake than nickel in a soil system. A large proportion of the nickel added to the soil system is fixed or complexed by the soil compounds (Traynor and Knezek 1973). More data on nickel toxicity to crops are quoted in the study by NAS/NAE (1973, p. 344). A nickel concentration of 0.5 mg/L was toxic to tomato seedlings, 0.5 to 5 mg/L to flax, 1 mg/L to hop plants and 2 mg/L to barley and beans. Leeks grown in treated soil had a nickel concentration of 7.0 mg/kg vs. a concentration of 2.0 mg/kg for leeks grown on an untreated soil. Similarly, the nickel concentration in globe beet tops was 16.5 mg/kg (control 3.2 mg/kg); globe beet roots, 13.0 mg/kg (control 1.65 mg/kg); potato roots, 0.57 mg/kg (control 0.25 mg/kg), etc. (LeRiche 1968).

Published data on the application of sewage sludge to agricultural soils showed that both the total and "extractable" nickel concentrations increased when the sludge's nickel concentration was high (Andersson and Nilsson 1972; Lund *et al.* 1976). When the sludge's nickel concentration was low, accumulation did not seem to occur. Soil irrigated with wastewater effluent containing a concentration of 0.050 mg Ni/L, and with a mixture of effluent and sludge containing an overall concentration of 0.075 mg Ni/L, over a long period (10 years) showed no significant increase

in extractable (with 0.1 *N* HCl) nickel concentrations over a control plot (Sidle *et al*. 1977).

Recommended Objective for Irrigation Waters

The above data show that for certain plants, nickel toxicity symptoms appear at plant "available" concentrations of approximately 0.5 to 1 mg/L in nutrient solutions and 1 mg/kg in soil. The data also showed that when irrigation waters come in contact with soil, part of the nickel is complexed and is mostly unavailable to plants, but the remainder of the nickel is generally available and can cause crop damage. At low concentrations, nickel does not seem to accumulate in soils.

NAS/NAE (1973, p. 344) has recommended a maximum nickel concentration in irrigation waters of 0.2 mg/L for continued use on all soils. For neutral fine-textured soils for a period of up to 20 years, 2.0 mg/L is recommended as the maximum limit. Pratt (1973) has suggested that on the fine-textured alkaline soils, concentrations of 2.0 mg/L might be used for 50 years and 5.0 mg/L might be used for 20 years without danger to plants.

Considering these data, maximum limits of 0.2 mg/L for total nickel in irrigation waters for continuous use and 2.0 mg/L for periods of up to 20 years or for intermittent or occasional use are recommended.

Recreation and Aesthetics

Recreational waters where there are cottages and permanent campsites should meet the same nickel requirement as that recommended for surface waters that are used as sources of drinking water, i.e., a maximum of 0.20 mg/L (assuming no treatment).

Nickel has not been shown to accumulate in biological tissue, so fish caught during sport fishing will not be a hazard to humans in relation to nickel content.

Where the recommended objective for aquatic life (0.025 mg/L in soft water and 0.25 mg/L in water of hardness >150 mg/L as $CaCO_3$) is in effect, the water will be safe for sports involving physical contact with the water and for drinking.

Recommended Objectives for Recreational Waters

The recommended objective for recreational waters is 0.2 mg/L.

Industrial Water Supplies

The only industry in which the concentration of nickel in process water is of importance is the food industry.

The general principle that the water which is incorporated in all processed food must meet the requirements for drinking water applies.

Recommended Objective for Industrial Water Supplies

A maximum level of 0.2 mg/L is recommended for water that is incorporated in the final product in the food industry. This includes water used for the cooling of cans in the canning industry.

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